A CLINICAL AND NEUROIMAGING STUDY

OF

CEREBRAL VENOUS THROMBOSIS.

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CERTIFICATE

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DECLARATION

I solemnly declare that the dissertation entitled ""**A CLINICAL AND NEUROIMAGING STUDY OF CEREBRAL VENOUS THROMBOSIS"** is done by me at Madras Medical College and Hospital, during 2004 - 2006 under the guidance and supervision of **Prof.V.K.RAJAMANI, M.D.** This dissertation is submitted to The Tamil Nadu Dr.M.G.R. Medical University towards the partial fulfilment of requirements for the award of **M.D. DEGREE IN GENERAL MEDICINE (BRANCH I)**.

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CONTENTS

INTRODUCTION

Thrombosis of cerebral veins and sinus is a distinct cerebrovascular disorder, unlike arterial stroke, most often affects young adults and children. CVT may be difficult to diagnose clinically because of its various and non - specific manifestations and multiple associated conditions and etiologies.

During the past decade, increased awareness of the diagnosis, improved neuroimaging like 3D - MR imaging techniques, and more effective treatment has improved the prognosis.

Blood from the brain drains, through small cerebral veins into larger veins such as the vein of Galen. These bigger veins empty into dural sinuses which themselves are drained mostly by the internal jugular veins. The venous territories are less well defined than are arterial territories due the presence of extensive anastomoses between cortical veins. These allow the development of collateral circulation in the event of an occlusion. The main cerebral venous sinuses affected by CVT are the superior sagittal sinus and lateral sinus.

Pathophysiologically, there are important differences between arterial and venous thrombosis. CVT has been described as a continuing process in which the balance of prothrombotic and thrombolytic process is disturbed leading to progression of the venous thrombus with time. This slow growth of the thrombus and the good collateralisation of the venous vessels probably explain the usually gradual onset of symptoms, frequently over weeks and months. Sudden onset, however has been described especially in post partum patients.

The symptoms and signs associated with CVT are relatively nonspecific. Headache is the presenting symptom in 70 - 90% of cases. Focal deficits such as hemiparesis and hemisensory disturbances, seizures, impairment of level of consciousness and papilledema are other clinical presentations. Some patients can present with a syndrome resembling isolated intracranial hypertension, with headache, papilledema and visual disturbances.. The onset may be acute, subacute or insidious, most patients presenting with symptoms that evolved over days or weeks.

CVT can occur on a spontaneous basis or secondary to a hypercoaguable state, dehydration, infection,malignancy or dural sinus compression. predisposing factors can be identified in upto 80% of patients. Numerous conditions can cause or predispose to CVT and more than one cause can be found in a particular patient.

A principal distinction can be made between infective and non - infective causes. Amongst the non - infective causes, systemic conditions such as connective tissue disease, other granulomatous or inflammatory disorders and malignancies are most common. In the middle East, Behcets disease may be responsible for upto 25% of cases.

In Young Women, CVT occurs more frequently during the puerperium than during pregnancy. Oral contraceptive and various coagulation disorders have been associated with increased risk of CVT. Hereditary prothrombotic conditions such as Factor V Leiden (Leading to increased resistance to activated protein C), deficiency of protein C and S, and antithrombin III as well

as prothrombin gene mutations may account for 10 - 15% of cases. The risk of a carrier of any of these prothrombotic conditions developing CVT in increased by the co-existence of other predisposing factors.

Investigations should focus in establishing the diagnosis and searching for underlying causes. CT, MRI, conventional angiography and more recently CT venography have been used to detect CVT. MRI conbined with MR venography has largely replaced invasive cerebral angiography and conventional CT. CT however remain the first imaging modality to be used to exclude other conditions such as intra cerebral hemorrhage and abcess. MRV in conjunction with conventional MRI can accurately diagnose CVT and is reliable as the sole investigation for this condition. Recently CT venography has been shown to have sensitivity equal to MRV in visualising thrombosed sinus or cerebral veins.

Intravenous heparin should be the first - line treatment, even in the presence of hemorrhagic infarction, provided there are no general contra - indications to its use. If the patient deteriorates despite heparinastion or presents with moribund with coma, selective catheter guided thrombolysis may be an option, inspite of the increased hemorrhagic risk. This should be followed by 3 - 6 months of oral anti-coagulation.

Mortality ranks between 5.5% and 18% in recent series. More than 80% of all patients have a good neurologic outcome.

AIM OF THE STUDY

The aim of the present study are

1. To study the clinical profile of patients with cerebral venous thrombosis.

2. To study the neuro imaging findings, risk factors and possible etiology, clinical course and outcome in patients with cerebral venous thrombosis.

REVIEW OF LITERATURE

BACKGROUND AND HISTORY

Cerebral vein and dural sinus thrombosis was first described in 1825 by Ribes[60] in a post mortem report. Since then many series have been published. However the true incidence of CVT is still unknown. In most of the autopsy series, it was found very low.

Thrombosis of cerebral veins & sinus in a distinct cerebrovascular disorder, that unlike arterial stroke affects young adults and children. The symptoms and clinical course are highly variable. The estimated incidence in 3 - 4 case / million population.

During past decade increased awareness of diagnosis, improved neuroimaging techniques and more effective treatment has improved the prognosis.

RELEVANT VENOUS ANATOMY

Blood from brain is drained by cerebral veins that empty into dural sinuses, themselves mostly drained by the internal jugular vein.

DURAL SINUSES

Superior Sagittal Sinus

SSS lies in the attached border of the falx cerebri. It starts at the foramen caecum and runs backward towards the occipital protuberance, where it joins

with the straight sinus & lateral sinus to form the torcular herophili. The anterior part is narrow or sometimes absent, replaced by two cerebral veins that join behind the coronal suture.

The SSS receives superficial cerebral veins and drains major part of the cortex. It also receives diploic veins, themselves connected to scalp veins by emissary veins which explain some cases of SSS thrombosis after cutaneous infections.

The SSS and other sinuses play a major role in CSF circulation because they contain most of the arachnoid villi and granulations in which most of the CSF absorption takes place. Thus there is a direct dependency of CSF pressure on intracranial venous pressure, accounting for the frequency on increased ICP with SSS or LS thrombosis.

LATERAL SIUSES

The LS extend from the torcular herophili to the jugular bulbs and consists of two portions :- the transverse portion, which is attached to the tentorium & the sigmoid portion, which runs on the inner aspect of mastoid process and in thereby susceptive to infective thormbosis in patients with mastoiditis or otitis media.

This LS drain blood from cerebellum, brainstem and posterior part of cerebral hemisphere. They also receive some diploic veins and some small vein from middle ear, yet another possible source of septic thrombosis. Numerous

LS anatomic variations may be misinterpreted as sinus occlusion in angiography. In particular, the right LS which often a direct continuation of SSS is frequently larger than the left which receives most of the blood from the straight sinus. In Hackers [43] study, the transverse portions were not visualised on ipsilateral carotid angiogram in 14% cases on left side and in 3.3% on right side whereas sigmoid portions which may be directly injected via cerebral veins, failed to fill 4% of cases in the left side and were always demostrated on the right. An isolated lack filling of left transverse sinus is thus more suggestive of hypoplasia than thrombosis.

CAVERNOUS SINUS

Cavernous sinus consists of trabeculated cavities formed by separation of layers of the dura & located on each side of the sella turcica, superolateral to the sphenoid air sinus. The oculomotor, trochlear cranial nerve, along with the ophthalmic and maxillary branches of the trigeminal nerve, course along the lateral wall of the cavernous sinuses, whereas the abducens nerve and carotid artery with its surrounding sympathetic fibres plexus are located within the centre of the sinus.

Cavernous sinuses drain the blood from the orbits through the opthalmic veins & from the anterior part of the base of the brain by sphenoparietal sinus and middle cerebral veins. They empty into both the superior and inferior petrosal sinus and ultimately into the internal jugular veins. In contrast to other varieties of sinus thrombosis, infection is still the leading cause of thrombosis.

CEREBRAL VEINS

The three groups of veins that drain the blood supply from the brain are superficial cerebral (or cortical), the deep cerebral veins of the posterior fossa.

SUPERIOR CEREBRAL VEINS

Some of the cortical veins, the frontal, parietal and occipital cerebral veins drain the cortex ascendingly into the SSS, whereas the middle cerebral vein drain descendingly into cavernous sinus. These veins are linked by Trolard great anastamotic vein which connects SSS to middle cerebral vein. The middle cerebral vein is connected to CS by Labbe's vein.

These cortical veins have thin walls, no muscle fibres and no valves, permitting both dilatation and reversal of the direction of blood flow when the sinus in which they drain is occluded. They are linked by numerous anastomoses allowing development of a collateral circulation, which probably explains the good prognosis of same CVT. The number and location of cortical are inconstant. This anatomical variability, together with the possibility of flow reversal and development of collateral circulation, accounts for the absence of well delineated venous territories and consequently of well defined clinical syndromes of cortical vein thrombosis.

DEEP CEREBRAL VEINS

Blood from deep white matter of cerebral hemispheres & from the basal ganglia is drained by the internal cerebral and basal veins that join to form the great vein of Galen which drains into straight sinus. Deep system is constant and always visualised at angiography, so that its thrombosis is easily recognised.

VEINS OF THE POSTERIOR FOSSA

They are divided into three groups: superior veins draining into the Galenic system, anterior veins drains into the petrosal sinus and posterior veins draining into the torcular & neighbouring SS and LS.

PATHOGENESIS

To understand the symptomss and signs of sinus thrombosis two different mechanisms should be distinguished.

1. Thrombosis of cerebral vein - with local effects caused by venous obstruction.

2. Thrombosis of the major sinus - which cause increased intracranial pressure.

I Occlusion of the cerebral vein causes localised edema of the brain and venous infarction. The latter can merge and become large hematomas which have characteristic appearance on CT scan. Two different kinds of cerebral edema can develop.

***Cytotoxic edema** caused by ischemia which damages energy dependent cellular pumps leading to cellular swelling.

***Vasogenic edema** caused by disruption in thee blood brain barrieer and leakage of blood plasma into interstitial space. Vasogenic edema in reversible if the condition is treated successfully.

II. Thrombosis of major sinus : The second mechanism is development of intracranial hypertension as a result of occlusion of the major venous sinus. Thrombosis of sinus leads to increased venous pressure, impaired absorption of CSF through subarachnoid villi and consequently increased intracranial pressure.

The obstruction to the drainage of CSF is located at the end of the transport pathway and no pressure gradient develops between subarachnoid spaces at the surface of the brain and ventricles. Hence ventricles do not dilate and hydrocephalus does not normally complicate sinus thrombosis. About 1/5th of patient with sinus thrombosis have intracranial hypertension only without signs of cortical venous thrombosis.

The international study on cerebral veins & dural venous thrombosis (ISCVT)[38], observed the following frequencies of thrombosis in various sinus:

Superior sagittal sinus	- 62%
Transverse sinus	- 86%
Vein of Galen & internal cerebral vein	- 11%
Jugular veins	- 12%
Cortical veins	- 17%

EPIDEMIOLOGY

The exact incidence of CVT, in still under debate because of scarcity of scientifically planned epidemiological studies in the available literature. Hospital data has been used to determine its prevalence in the community.

Kalbag & Woolf et al[46] indicated that CVT was the principal cause of death in only 21.7 persons/year in England & Wales between 1952 & 1961.

Towbin et al[72] found in 9% of 182 consecutive autopsies insists that primary CVT in adults in still under recognised disease.

On the other hand in studies Nagaraja et al[52], Chopra et al[22] & Srinivasan et al[66], aseptic CVT occuring in pregnancy and puerperium has been reported very frequently from the Indian subcontinent and CVTconstitutes 10 - 15% of stroke in the young and was commonest cause of stroke in premenopausal women.

Srinivasan et al[60] encountered 50 cases of severe CVT amongst 1000 deliveries performed per year. It has been estimated the prevalence rate in developing countries in approximately 10 times more than the developed countries.

The more recent publication of large scale series, suggest that over the past 5 to 10 years CVT has been diagnosed more frequently due to greater awareness and availability of better non-invasive diagnostic techniques. CVT is slightly more common in women particularly in the age group of 20 to 35 due to pregnancy, puerperium & OCP use. Mean age in most large studies was between 37 & 38 years though all ages can be affected.

CAUSES AND RISK FACTORS

Predisposing factors can be identified in upto 80% of patients (Bousser et al)[9]. Numerous conditions can cause or predispose to CVT and often more than one cause will be found in an individual. A prinicipal distinction can be made between infection and non-infectious causes.

In Bousser et al.,[9] infective cases have declined and were responsible only for 8% of causes. Cavernous sinus thrombosis remains the most common form of septic thrombosis usually following staphylococcal infections of middle third of the face. Other infections include sphenoid or ethmoid sinusitis, dental abscess and less often otitis media.

Among the non-infective causes, CVT occurs frequently during puerperium than in late pregnancy and remains very common in developing countries whereas in the developed countries the role of OCP is more important.

De Bruijn et al.,[27,29] found in age adjusted odds ratio of 13 for OCP use and risk of CVT. Hereditary prothombotic conditions such as Factor V Leiden (leading to increased resistance to activated protein C), deficiency of protein C, protein S and antithrombin deficiency as well as prothrombin gene mutations account for 10 - 15% of case of CVT. The detection of congenital thrombophilia should be systematic in CVT since it potentiates the risk of venous thrombosis associated with other condition including OCP or puerperium.

Other causes include connective tissue disorders, granulomatous & malignancies. Daif et al.,[25] in study of 40 cases from Saudi Arabia found that in the Middle East, Behcet's disease may be responsible in 25% of cases.

The etiology remains unknown in 20 - 35% of cases. Finding etiology for CVT necessitates an extensive initial working and when no cause in found, a long term follow up with repeated investigations. In some cases initially interpreted as idiopathic, a general disease can be discovered some months later.

CAUSES & PREDISPOSING CONDITIONS[68]

I. Infectious causes

LOCAL

Otitis, mastoiditis and sinusitis

Meningitis, intracranial abscess

Direct septic trauma

GENERAL

Systemic infectious disease (Bacterial, viral, fungal and parasitic)

II. NON-INFECTIOUS CAUSES

GENETIC PROTHROMBOTIC CONDITIONS

Antithrombin III deficiency

Protein C & S deficiency

Factor V Leiden mutation

Prothrombin mutation (20210)

Homocysteinemia

ACQUIRED PROTHROMBOTIC STATES

Nephrotic syndrome

Antiphospholipid antibodies

Homocysteinemia

Pregnancy

Puerperium

INFLAMMATORY DISEASES

SLE

Behcet's disease

Inflammatory bowel disease

Wegener's granulomatosis

Sarcoidosis

HEMATOLOGIC CONDITIONS

Polycythemia (primary & secondary)

Thrombocythemia

Leukemia

Anemia

DRUGS

OCP

Asparaginase

MECHANICAL CAUSES

Head injury

Injury to sinus or jugular vein, jugular vein catheterization

Neurosurgical procedures

Lumbar puncture

MISCELLANOUS

Dehydration especially children

Malignancies

CLINICAL MANIFESTATIONS

CVT presents with a wide spectrum of symptoms and signs:

* In Bousser et al[9] series, headache is the presenting symptom in 70 - 90% of cases.

  - the earliest symptom in two third of cases.

  - it has no specific features.

  - its mostly diffuse, progressive and permanent but it can misleading, minicking migraine or headache of SAH.

* Focal deficits such as hemiparesis & hemisensory disturbances, seizures, impairment of consciousness and papilledema occurs in one third to three quarters of cases. Other signs such as cerebellar incoordination or psychiatric disturbances can occur.

* Seizures occur in 40% of cases, a far higher percentage than arterial stroke. Seizures are limited and focal in 50% of these patients but may generalise to life threatening status epilepticus.

Thrombosis of deep venous system - the straight sinus & its branches - causes centrally located often bilateral thalamic lesions with behavioural symptoms such as delirium, amnesia and mutism which may be the only manifestation of sinus thrombosis.

Causes of coma in CVT include brainstem compression due to the infarct/hemorrhage, seizures and involvement of thalamus. Patients with increased intracranial tension have headache but no neurologic symptoms with exception of diplopia due to involvement of sixth nerve when the intracranial pressure is high.

The mode of onset of symptoms is highly variable. In Bousser MG[12] et al series of 135 patients it was acute (<48hrs) in 37 patients (27%), subacute (>48 hrs to <30 days) in 67 patients (50%) and chronic (>30 days in 31 patients (23%).

DIAGNOSIS

Although the clinical presentation is highly variable, diagnosis of CVT should be considered in young and middle aged patients with

i) Recent unusual headache or stroke like symptoms in the absence of usual vascular risk factors.

ii) In patients with intracranial hypertension and

iii) In patients with CT evidence of hemorrhagic infarcts especially if the infarcts are multiple and not confined to arterial vascular territories.

The average delay from the on set of symptoms to diagnosis is seven days.

INVESTIGATIONS

CVT is an uncommon condition and hence unless this condition is suspected prior to embarking on investigations the diagnosis is likely to be missed. Therefore, it is reasonable to entertain this diagnostic possibility if the circumstances are conducive to development of CVT.

1. COMPUTED TOMOGRAPHY

CT scan with and without contrast injection is the first neuroimaging examination carried out in patients with headache, focal deficits or seizures particularly on an emergency basis. CT scan is extremely useful to rule out many of the conditions that CVT mimics. It can occassionally detect the lesions that can themselves cause CVT such meningiomas, abscess, sinusitis & mastoiditis.

CT findings can be divided into direct and indirect signs.

DIRECT SIGNS OF CVT

a. Cord sign

- visible on unenhanced CT scan represents the spontaneous visualisation of thrombosed cortical veins.

According to Bousser et al[12], it is extremely rare in his series (1 in 116 cases of his series). It is also seen in internal cerebral vein and vein of Galen thrombosis.

b. Dense triangle sign

- Reflects spontaneous SSS opacification by freshly coagulated blood.

It was present in less than 2% of case of Bousser et al[12].

c. Empty delta sign

- was described by Buonanno et al[15], appears after contrast injection & reflects the opacification of collateral veins in the SSS contrasting with non-injection of clot inside the sinus.

It is the most frequent direct sign approximately 35% of published cases.

Indirect signs of CVT

- Intense contrast enhancement of falx and tentorium. Tentorial enhancement is usually thought due to straight sinus thrombosis.
- Small ventricular system
- Reduction of sulcal pattern due to cerebral edema
- White matter hypodensity without contrast enhancement suggestive of cerebral edema (present upto 75% of cases in study by Ford et al.,[41])

- Hemorrhagic, venous infarcts in 10 - 50 % of the cases. In rare instances there is associated subarachnoid hemorrhage or subdural hematoma.

- Non hemorrhagic venous infarcts are also frequent.

The place of CT scan in the diagnostic strategy of CVT in mainly to rule out other conditions such as arterial stroke, abscess, tumors and SAH on emergency basis. In 10 - 20% cases, CT is normal in patient proven CVT. In a minority of cases it will show direct pathognomic signs of CVT but more frequently only indirect signs are present and MRI or angiographic information should be obtained.

Recently CT venography has been shown to have sensitivity equal to MRV in visualising thrombosed sinus or cerebral veins.

2. MAGNETIC RESONANCE IMAGING

MRI offers major advantages for the evaluation of possible CVT - sensitivity to blood flow, ability to visualise the thrombus itself and non invasiveness. A variety of MR findings have been described, mainly relating to the evaluation of thrombosis, which evolve over time.

1. **In acute phase -** absence of normal venous flow void in T1 & T2 weighted images. Thrombus itself appears isotense in T1 & hypointense in T2 weighted images.

2. **During 5 - 15 days,** absence of flow void persists but the thrombus becomes hyperintensive initially the thrombus becomes

hyperintense intially in T1 & then on T2 weighted images. In large vessels, these changes start in the periphery and proceed towards the centre. They represent the aging of the thrombus with biochemical conversion of oxyhemoglobin to methhemoglobin.

This intermediate pattern (increased signal on T1 & T2 weighted images) is diagnostic of CVT and is by far the most frequent.

3. Late changes (approximately 2 to 4 weeks after onset)

Can reveal the beginning of vascular recanalisation with resumption of flow void in the previously thrombosed vessels.

However at 6 months, more than two third of cases still have some heterogenous localised signal abnormalities which can persist for years and should not be mistaken for recurrent acute CVT. In some cases, however, interpretation of MRI images is not easy because of false-negative and false-positive images.

The combination of non-contrast MRI plus MRA is now-a-days the best method for the diagnosis & follow-up of CVT. It should be performed as first investigation in case of high clinical suspicion. Its use is limited in certain situations such as deeply comatose subjects requiring artificial ventilation.

OTHER NEUROLOGIC INVESTIGATIONS

3. ANGIOGRAPHY

IA angiography ± DSA provides high definition images but is associated with small (<5%) risk of neurological complications. Filling deficits within dural sinus or their complete occlusion demonstrated an delayed venous phase projections. It can also demonstrate appearance of deep and superficial cortical collateral veins (cork-screw veins).

4. LUMBAR PUNCTURE

- To exclude other pathologies such as meningitis and SAH.
- CSF in CVT may be normally or there may be an increase in protein, white cells, red cells alone or in combination.
- Measurement of CSF pressure may be useful in establishing a baseline & also as a therapeutic measure if vision is threatened.

5. GENERAL INVESTIGATION

After CVT has been established, investigations should be directed toward demonstrating the underlying etiology. Because of multiplicity of etiologies, this is a long & difficult task whenever the cause is not clinically evident. Fever, increased ESR or PMN points to infective, inflammatory or malignant cause.

Investigations for hereditary thromboophilic conditions like factor V Leiden mutation, protein C and protein S, deficiency, antithrombin deficiency, homocysteinemia, should be done systemically looked for in patients with CVT, whether or not other potential causes are present, because the risk of CVT in patients carrying any of these prothrombotic conditions, is increased by co-existence of other predisposing factors.

Investigations for connective tissue disorders like ANA, APLA, Behcet's may be done in suspected cases.

TREATMENT

General Measures

The combination of acutely increased intracranial pressure & large venous infarcts is dangerous and patient die within hours from cerebral herniation. The priority of treatment in the acute phase is to stabilise the patients condition and to prevent or reverse cerebral herniation.

This may require the administration of IV mannitol, surgical removal of the hemorrhagic infarct or decompressive hemicraniotomy. It is not known whether administration of corticosteroids in the acute phase improves outcome. Possible causes of sinus thrombosis such as infections, connective tissue disorders and malignancies should be searched for and treated. Antibiotic treatment very important in CVT due to infectious causes.

Symptomatic treatment, like anticonvulsants are very important in patients who present with seizure. The question of duration of treatment with anticonvulsants remain open. In Bousser et al.,[12] anticonvulsants were progressively discontinued 1 year after CVT in patients with normal EEG and no recurrent seizures.

Anticoagulation

The most obvious treatment option in anticoagulation with heparin to arrest the thrombotic process & to prevent pulmonary emboli which may complicate sinus thrombosis. However, anticoagulation treatment has raised much controversy because of the tendency of venous infarcts to become hemorrhagic and about 40% of all patients with sinus thrombosis have hemorrhagic infarct even before anticoagulation is started.

A study by Einhaupl et al.,[36] showed significant benefit with heparin even patients who had hemorrhagic infarcts. Diaz et al.,[33] reviewed 203 CVT cases reported between 1942 & 1990 and compared the outcome of patients treated and not treated (149) with heparin. 91% survived in the first group compared with 36% in the second group.

Nagaraja et al.,[54] compared the effect of IV un-fractionated heparin with that of placebo in 57 women from India who had puerperal sinus thrombosis which showed a non-significant benefit of anticoagulant treatment as compared with placebo. There is thus good evidence that the benefit / risk ratio of heparin

is favourable in patients with CVT, but there are still disagreements on the best indications.

Most neurologist now start treatment with heparin as soon as this diagnosis of CVT in confirmed even in the presence of hemorrhagic infarcts unless contra-indication exists for their general use. There are no studies that compared the effect of un-fractionated heparin in treatment with sinus thrombosis. There is no fixed duration for heparin treatment but empirically it is prolonged until the patient improves or atleast stabilises.

It is then replaced with oral anticoagulants to obtain an INR between 2.5 and 3.5. Usually vitamin K antagonists are given are six months after 1st episode of sinus thrombosis. But prolonged treatment is warranted whenever prothombotic situation exists such as malignancy, inflammatory disease (Behcet's disease and SLE), inherited thrombophilia or history of recurrent venous thromboses.

Thrombolytics

Anticoagulants are widely used as first line therapy, their rationale being to avoid thrombus extension and favour spontaneous thrombus dissolution. Thrombolysis is considered when a rapid recanalisation is sought especially in patients who deteriorate despite anticoagulant therapy.

OUTCOME

SHORT TERM OUTCOME:

Mortality

There are three main causes of death in CVT - the brain lesion itself, particularly when massive hemorrhagic infarct is present, intercurrent complications such as sepsis, uncontrolled seizures and pulmonary embolism. In the series by Bousser MG et al.,[9] Einhaupl et al.,[36] DeBruijn et al.,[30] the mortality ranks between 5.5% and 18%.

Factors classically considered to suggest a bad prognosis are the rate of:

- rapid evolution of thrombus.
- age of the patient (with high mortality in infants & aged).
- infectious etiology.
- focal symptoms and coma.
- presence of hemorrhagic infarct.
- involvement of deep cerebral and cerebellar vein thrombosis.

DeBruijn et al[30]., a study of prospective series of 59 patients identified that following factors related to poor outcome after 12 weeks,

* Papilledema,
* Altered sensorium,
* Coma,
* Age older than 33 years,
* Diagnostic delay < = 10 days,
* Intracerebral hemorrhage,
* Involvement of straight sinus.

Isolated intracranial hypertension and delta sign on CT were associated with good outcome.

FUNCTIONAL RECOVERY AND LONG TERM OUTCOME

It has long been recognised that if survival occurs in CVT, the prognosis for recovery of function is much better than in arterial thrombosis. A minority (15 to 20%) of patients are left with disabling sequelae such as focal deficits seizures, optic atrophy. In a study of 79 patients by Stolz E et al.,[70] most frequent complications on long term follow-up was epilepsy and recurrent venous thrombosis.

According to Preter et al.,[58] on follow-up of 77 patients residual epilepsy was reported in 10 - 30% of the patients and recurrence was seen in 11.7% of patients.

MATERIALS AND METHODS

PATIENTS AND METHODS

The subjects for the present study comprised of patients admitted in Government General Hospital in Medical and Neurology Wards, Chennai, during the period of September 2003 and January 2006. (About 47 patients of age between 15 - 70 years were studied).

INCLUSION CRITERIA

The subjects were diagnosed to have CVT and included in the study when an appropriate clinical picture of the patient was supported by one or more of the following.

1. CT Scan - showing unequivocal direct or indirect evidence of cerebral venous thrombosis.

2. MRI showing evidence of cerebral venous thrombosis.

    a. Partial can complete absence of filling of one or more dural sinus.

    b. Visualisation of thrombus $\pm$

    c. Parenchymal lesions suggestion of CVT.

3. Age of the patient >14 years.

A total of 47 patients (24 men and 23 women) fulfilling the above criteria were studied.

* A detailed clinical history was taken with special emphasis on the suspected precipitating or predisposing factors dehydration, fever, sepsis, anemia, parity, underlying medical illness, obstetric history, joint pains, oral / genital ulcers, abortions, oral contraception.

* Detailed examination of the patients was carried out including :

  a. General physical examination - any evidence of anemia, dehydration, sepsis, deep vein thrombosis of leg or arthritis was noted.

  b. Cardiovascular, respiratory and per abdominal examination was carried out to look for any evidence of systemic involvement.

  c. A detailed neurological examination was done which included (i) assessment of level of sensorium (ii) any evidence of raised intracranial tension and iii) any focal deficits or bilateral neurological signs and iv) any evidence of meningeal limitation.

* All the patients were investigated with hemogram, ESR, Total and differenital leucocyte counts, platelet count, blood sugar,

Blood urea, serum creatinine, lipid profile, bleeding time, clotting time, prothrombin time, urine analysis, chest x-ray.

All the patients underwent CT scan of head plain and with contrast enhancement.

Also all the patient underwent MRI imaging of brain with MR angiogram (venogram).

CSF analysis was done any in 3 patients with meningeal signs..

Other ancillary investigations, protein C, protein S APLA, ANA, anti - thrombim III were performed only in few patients with high clinical suspician.

The results were analysed.

OBSERVATIONS AND RESULTS

I. AGE AND SEX DISTRIBUTION

Table - 1A : Age Distribution

Age	No. of Patients	Percentage
15 - 20	2	4%
21 - 30	26	55%
31 - 40	15	31%
41 - 50	1	2%
51 - 60	2	4%
61 - 70	1	2%

Mean age of the total cohort - 28.5 years (Range 15 to 70 years).

Table I B : Sex Distribution

Sex	No. of Patients	Percentage
Males	24	51%
Females	23	49%

There were 24 men (51%) and 23 women (49%) in the study.

II. AGE DISTRIBUTION BETWEEN PUERPERAL AND NON - PUERPERNAL GROUPS.

	No. of Patients	Percentage	Mean Age
Puerperal	14	29.7%	25.1 years
Non Puerperal	33	60.8%	31.7 years

Of the 47 patients 14 patients (29.7%) were puerperal. Most of the patients (60.8%) were cases of non - puerperal CVT.

Mean age of puerperal CVT - 25.1 years.

Mean age of non - puerperal CVT - 31.7 years

Patients with puerperal CVT were of relatively younger age.

III. MODE OF ONSET IN TOTAL COHORT AND PUERPERAL CVT PATIENTS.

Table III shows mode of onset of the symptoms. Those who presented within 48 hours were considered to have acute onset; with onset longer than 48 hrs but less than 1 month as sub - acute, and with onset more than one month as Chronic (Bousser et al.,[9]).

TABLE IIIA - MODE OF ONSET IN TOTAL COHORTS

	No. of Patients(47)	**Percentage**
Acute	11	23.4%
Subacute	33	70.2%
Chronic	3	64%

Most of the patients had a sub-acute presentation.

11 (23.4%) had acute, 33 (70.2%) had sub-acute and 3(6.4%) had chronic onset.

TABLE IIIB - MODE OF ONSET IN PUERPERAL AND NON PUERPERAL CVT PATIENTS

	Puerperal (n = 14)	**Non Puerperal (n = 33)**
Acute	8 (57%)	3(9.1%)
Subacute	6 (43%)	27(81.8%)
Chronic	-	3(9.1%)

Most of the puerperal CVT patients had acute onset of presentation. 8(57%) had acute onset and 6(43%) had subacute onset. No patients with puerperal CVT had chronic onset of symptoms.

Most of the non - puerperal CVT had subacute onset of presentation. 3(9.1%) had acute onset, 27(81.8%) had sub-acute onset and 3(9.1%) had chronic onset.

TABLE IVA - CLINICAL FEATURES

	No. of Patients (n=47)	**Percentage**
Symptoms		
Headache	40	85%
Seizures	19	40.4%
Alteration on level of consciousness	22	46.8%
Signs		
Focal Deficits	26	55.3%
i. Sensory	1	2%
ii. Motor		
* Hemiparesis / Hemiplegia	15	31.9%
* Monoparesis / Monoplegia	5	10.6%
* Paraplegia	1	2%
* Multiple cranial nerve deficits	4	8.5%
Bilateral papilledema	37	78.7%
Meningeal Irritation signs	3	6.3%
Isolated ICT	13	27.6%

Headache was the most common presenting system. It was seen in 40(85.1%) cases.

Seizures were another common symptom at presentation. 19 patients (40.4%) of all cases had seizures.

Table IVB give the distribution of type of seizures in each group.

TABLE - IVB : TYPE OF SEIZURES

	No. of Patients (n=19)	**Percentage**
Focal	2	10.5%
Focal with secondary generalisation	1	5.2%
GTCS	13	68.4%
Status epilepticus	3	15.7%

68.4% of the cases with seizures were generalised term clonic seizures. While 10.5% had focal seizures, 5.2% had focal seizures with secondary generalisation. 15.7% presented with status eplilepticus.

TABLE - IVC : LEVEL OF CONSIOUSNESS AT PRESENTATION

	No. of Patients (n=47)	**Percentage**
Consious	25	54.2%
Altered level of consiousness	22	46.8%
Drowsy	10	21.2%
Stupor	8	17%
Coma	4	8.5%

22 patients (46.8%) had alteration in consiouness level at presentation. 4 patients were comatose at presentation.

At presentation bilateral papilledema as present in 37 patients (78.7%).

13 patients (27.6%) presented with features isolated intracranial hypertension.

Focal deficits were present in 26 (55.3%). Hemiparesis was the common focal deficit in 15(57.6). 5(19.2%) had monoparesis (faciobrachial monoparesis), 1(3%) had paraplegia, 4(15.3%) had multiple cranial nerve deficits, 1(2%) had sensory disturbance.

TABLE V - CT SCAN FIDNINGS

	No. of Patients (n=47)	**Percentage**
Normal	10	21.2%
Non - hemorrhagic infarct	9	19.1%
Hemorrhagic infarcts	17	36.1%
Intrancerebal hematoma	-	-
Sub-arachnoid hemorrhage	3	6.2%
Sub-dural hematoma	1	2%
Direct Signs		
Cord Sign	4	8.5%
Empty delta sign	3	6.3%
Indirect signs		
Cerebral edema	5	10.6%
Obliteration of cistern	5	4.2%
Ventricle size		
a. Squashed	4	8.5%
Tentorial enhancement	2	4.2%
Gyral enhancement	-	-
Falx enhancement	-	-

CT brain was normal in 10 (21%) patients. Hemorrhagic infarcts were most common parenchymal lesions. 17 patients (36.1%) had hemorrhagic infarcts. 9(19%) had non - haemorrhagic infarcts, 3(6.3%) had sub-arachnoid hemorrhage and 1(2%) had sub-dural hematoma.

Direct signs of CVT was present in 7(14.8%) patients. Cord sign was present in 4 (8.5%) patients. 3 (6.3%) had empty delta sign.

Indirect signs were common than directs signs.

16 patients (27.6%) had indirect signs of CVT like cerebral edema, tentorial enhancement obliteration of cisterns, squashing of ventricles.

TABLE VI : MRI FINDINGS

	No. of Patients (n=47)	**Percentage**
Sinus Involvement		
Superior sagittal sinus	35	74.7%
Inferior sagital sinus	1	2%
Transverse sinus	16	34%
Sigmoid sinus	17	36.1%
Cavernous sinus	2	4.2%
Straight sinus	6	12.7%
Internal jugular veins	2	4.2%
Cortical vein thrombosis	5	10.6%
Deep cerebral vein involvement	3	6.3%
Parenchymal lesions		
Non hemorrhagic infarct	9	19.1%
Hemorrhagic infarct	19	36.1%
Sub-arachnoid hemorrhage	3	6.3%
Sub dural hematoma	1	2%

TABLE - VIB

	No. of Patients (n=47)	**Percentage**
Isolated sinus	18	38.2%
- SSS	16	
- Isolated cortical vein	2	
Multiple sinus	29	61.7%

MRI with MR angiogram was done in all 47 patients studied. It was abnormal in all the patients.

Superior sagittal sinus was the most frequent sinus involved in CVT. 35 (74.7%) had partial or complete occlusion of superior sagittal sinus. It was the only sinus involved in 16(34%) of patients and associated with other sinus involvement in 19 patients.

Sigmoid sinus and lateral sinus was the next commonly involved sinus. 17(36.1%) cases had sigmoid sinus thrombosis, 16(34%) had lateral sinus involvement. Other sinus were less frequently involved straight sinus in 6(12.7%), cavernous sinus in 2 (4.2%). Internal jugular thrombosis was seen in 4(8.5%), one patient had inferior sagitial sinus, 5 (10.6%) had cortical vein thrombosis and 3(6.3%) had deep cerebral venous involvement.

LP done in 3 patients with meningeal signs revealed mild elevation of proteins in two patients. In one patient TBM wad diagnosed.

TABLE - VII PREDIPOSING / ETIOLOGY FACTOR IDENTIFICATION.

	No. of Patients (n=47)	**Percentage**
Infectious etiology	7	14.5%
Mastoiditis	4	8.5%
Sinusitis	1	2%
TBM	1	2%
Systemic - HIV	1	2%
Non Infectious etiology	24	
Pueperium	14	29.7%
APLA	2	4.2%
Protein C deficiency	1	2%
Protein S deficiency	1	2%
OCP use	4	8.5%
SLE	2	4.2%
Unidentified	16	34%

Predisposing / etiologic factors were identified in 31(66%) of patients.

In 16 (34%) risk factors could not be identified.

There were varied risk factors identified in our patients.

Infections were identified as risk factors in 7(14.5%) cases. In 4(8.5%) mastoiditis, 1 (2%) sinusitis, 1(2%) TB meningitis, 1(2%) HIV were identified as risk factor.

Among the non - infectious causes, puerperium was the common predisposing factor 14(29.7%) were cases of puerperal CVT.

Among the non - puerperal female CVT patients 4(8.5%) were associated with OCP usage, 2(4.2%) were diagnosed as SLE. One of the SLE patient had post partum CVT and other patient had OCP use as additional risk.

Hereditary thrombophilic conditions were not extensively investigated due to financial, operational difficulties. Primary antiphospholipid antibody syndrome was identifical as risk factor in 2(4.2%) patients. Both were males. 1(2%) patient had both protein C and Protein S deficiency.

All the patients in our study were treated with anti - coagulants, anti - edema measures. Anti - convulsants were exhibited in patients who presented with seizures.

TABLE VIII - OUT COME AT 4 WEEKS

	No. of Patients (n=47)	**Percentage**
Death	2	4.2%
Complete recovery	34	72%
Residual deficit	11	23.4%

In our study 2 patients (4.2%) died. Both these patients had deep coma at presentation. At the end of 4 weeks, 34(72%) recovered completely, 11(23.4%) had neurological sequelae in the form of residual focal deficits (10 cases), seizures (2 patients), visual loss (cortical blindness) in one patient.

DISCUSSION

The first description of CVT appeared in French Literature in 1825 by Ribes[60] as a post mortem report. Since then many series have been published. However the exact incidence of CVT in still unknown.

Most of the initial reports were from autopsy material and it was found to be extremely low. Ehlers[35] found only 16 SSS thrombosis cases in a series of 12,500 autopsies, and Barnett[19] reported only 39 non - infective CVT in 20 years. Kalbag and Woolf[46] indicated that CVT was the principal cause of death in on only 1 per 2 million (21.7 persons per year) in England and Wales between 1952 and 1961. By contrast Towbin[72] found CVT in 9% of 182 consecutive autopsies. At Nimhans, Bangalore, of the 1710 brains examined 75 were of primary CVT (Nagaraja et al.,[52]).

The recent publication of large clinical series suggests that the true incidence in much higher than that thought from initial autopsy series (Krayenbuhl et al.,[48] Bansal et al.,[6] Ford et al.,[41] Rao et al.,[59]).

It is note worthy that 50% of all strokes in women occur in relation to pregnancy and puerperium. Cross et al.,[21] attributed majority of strokes during pregnancy and puerperium to arterial lesions. However, barring few exceptions, survey of Indian literature reveals, CVT is the commonest cause of stroke in puerperium. Srinivasan et al.,[63] reported an incidence of 4.5 per 1000 deliveries out of 8000 deliveries conducted in a year at a Government Hospital, Madurai.

Bousser et al.,[9] reported 38 patients of angiographically proven CVT in 8 years in a single neurology department. Cantu et al.,[17] reported 113 patients of CVT seen in their hospital in 20 years. Daif et al.,[25] had encountered 40 patients of out over 9 years in two of Saudi Arabias' Main Hospitals. Deschiens et al.,[32] reported that 110 patients of CVT were diagnosed between 1975 and 1990 in their hospital (France).

Though our study of 47 patients does not give precise information about the real incidence of the disease, the fact that they were seen in 2½ years at Government General Hospital, Chennai, suggests that CVT is not uncommon.

TABLE - VIII : MEAN AGE OF ONSET

Sl.No.	Authors	Mean age of onset (Years)
1.	Bousser et al.,[9]	40.8
2.	Daif et al.,[25]	27.8
3.	Deschiens et al.,[32]	36.2
4.	Zhang et al.,[75]	31
5.	De Bruijn et al.,[30]	37
6.	Terazzi et al.,[71]	44.8
7.	Present study	28.5

The mean age of onset of CVT in our study is similar to studies by Daif et al.,[25] Zhang et al[75]. However Bousser et al.,[9] Deschiens et al.,[32] De

Bruijn et al., [30] Terazzi et al.,[71] found higher mean age of onset. Most of their cases were non puerperal.

TABLE IX : AGE DISTRIBUTION

Sl.No.	Author (No. of Patients)	Age (Years)			
		15 - 20	**21 - 30**	**31 - 40**	**>40**
1.	Caroll et al.,[18] (Puerperal)	1 (25%)	2 (50%)	1 (25%)	-
2.	Bansal et al.,[6] (138) (Puerperal)	22 (15.9%)	90 (65.2%)	26 (18.8%)	-
3.	Bousser et al.,[9] (38) (all cases)	2 (5.3%)	10 (26.3%)	7 (18.4%)	19 (50%)
4.	Deschiens et al.,[32] (40) (all cases)	2 (5%)	11 (27.5%)	15 (37.5%)	12 (30%)
5.	Present study (47) (all cases)	2 (4%)	26 (55%)	15 (31%)	4 (8%)

The age distribution in our study is similar to most other studies. About 59% of the patients in our series had onset between 15 and 30 years of age. However 50% of cases of Bousser et al.,[9] series and 30% of Deschiens et al[32]. Patients were more than 40 years of age. In our series only 8% were more than 40 years of age.

TABLE X : SEX DISTRIBUTION

Sl.No.	Authors (No. of Patients)	Males	Females
1.	Bousser et al.,[9] (38)	21 (55.3%)	17 (44.7%)
2.	Cantu et al.,[17] (113)	13 (11.5%)	100 (88.5%)
3.	Daif et al.,[25] (40)	20 (50%)	20 (50%)
4.	Deschiens et al.,[32] (40)	10 (25%)	30 (75%)
5.	Zhang et al.,[75] (23)	9 (39.1%)	14 (60.9%)
6.	De Bruijn et al.,[30] (59)	9 (16.3%)	50 (84.7%)
7.	Terazzi et al.,[71] (48)	10 (30.9%)	38 (79.1%)
8.	Present Study	24 (51%)	23 (49%)

In our study the sex distribution is almost similar 51% males, 49% females. This is similar to the experience in Bousser et al.,[9] and Daif et al.,'s[25] series, where there were 55% and 50% males respectively. Most of other studies have reported an increased female incidence.

PUERPERAL AND NON PUERPERAL CVT

About 14(29%) of our patients were puerperal CVT and 33 (71%) were non - puerperal.

Only 1(2.6%) of the 38 patients of Bousser et al.,[9] was puerperal, while in Deschiens et al.,[32] study only 8(20%) of patients were puerperal. Only 1 (2.5%) of Daif et al.,[25] was post partum. 4 (21.1%) of 19 patients of Zuber et

al.,[74] related to pregnancy and puerperium. Similar to these studies, most of the cases in our study were non - puerperal.

This is in contrast to the previous Indian studies, in which majority of the patients were related to pregnancy and puerperium.

D.Nagaraja et al.,[52] had found that 200 out of the 230 patients (86%) of CVT, seen over 8 years were puerperal in nature.

Puerperal CVT was reported to be responsible for 25% of maternal deaths in Indian and complicate 4.5% of 1000 obstetric admissions Srinivasan et al[63]. Our study differs from the experience of other authors from India.

Cantu and Barinagarrementeria et al.,[17] study of 67 patients of CVT associated with pregnancy and puerperium, observed that CVT patients associated with pregnancy and puerperium, were younger compared to non - puerperal CVT, and had more acute onset of symptoms (82% Vs 54% in non - puerperal group).

In our study mean age of onset was 25.1 years in puerperal CVT and 31.7 years for non - puerperal cases. 57% of puerperal CVT had acute mode of presentation and 43% had subacute onset. On the other had 9% of non - puerperal CVT had acute onset. Most of the patients had sub-acute onset (81%) and 10% had chronic onset. Our study co-relates with cantu and Barrinagmenteria et al.,[17] experience.

TABLE XI : MODE OF ONSET

Sl. No.	Authors (No. of Patients)	Acute (48 hrs)	Subacute (>48 hrs < 1month)	Chronic (>1 month)
1.	Daif et al.,[25] (40)	14 (35%)	16 (40%)	10 (25%)
2.	Zhang et al.,[75] (23)	4 (17%)	11 (47%)	8 (34%)
3.	Terazzi et al.,[71] (48)	21 (44%)	17 (35%)	10 (21%)
4.	ISCVT[38] (624)	232 (37.2%)	346 (55.5%)	45 (7.2%)
5.	Present Study (47)	11 (23.4%)	33 (70.2%)	3 (6%)

About 70% of the patients in our study had a sub-acute onset of presentation. 55.5% of patients of ISCVT study, 40% of Daif et al.,[25] series had subacute onset. The mode of onset in our study in similar to most other studies.

The neurological symptoms and signs encountered in our study were those classically associated with CVT like, headache, seizures, alteration in conscious level, focal deficits, bilateral papilledema and isolated inhaeramial hypertension.

Headache - headache appears to be the most common and often the earliest symptom in CVT patients.

TABLE XIII HEADACHE IN VARIOUS SERIES

Sl.No.	Authors (No. of patients)	Percentage
1.	Prakash and Singla[57] (210)	57%
2	Krayen Buhl[48] (73)	63%
3.	Kalbag and Woolf[46] (34)	29%
4.	Bousser et al.,[9] (38)	74%
5.	Nagaraja[52], (200)	57%
6.	Ameri A & Bousser[3], (110)	75%
7.	Cantu,[17] (113)	80.5%
8.	Daif et al.,[25] (40)	82%
9.	Zhang, et al.,[75] (23)	100%
10.	Present Study (47)	85.1%

85.1% of our cases had headache. Thus similar to other studies, headache in the most frequent symptom in our study also.

SEIZURES

The manifestation that indicate cerebral cortical involvement are convulsions and paralysis. At times, seizures are the heralding symptoms and arouse the suspicion of the diagnosis.

Caroll et al.,[18] found seizures in 29.8% of 181 cases and that focal seizures were commonest. K.Srinivasan et al.,[64] found seizures in 68% of 135 cases and generalised seizures were common.

Nagaraja et al[52]., found generalised seizures in 35% and focal seizures in 23% of their 200 puerperal CVT patients. 14% of their patients had status epilepticus. Nagaraja et al[55]., (1997) reported seizures in 70% of their 405 patients of puerperal CVT (40% had generalised and 30% focal becoming generalised).

Bousser et al[9]., reported seizures in 29% of their 38 patients. Daif et al[25]., noted 10% of their 40 patients had seizures. Deschiens et al[32]., reported seizures in 40% of their patients.

In our study, 40.4% of the patients had seizures. Generalised seizures were the commonest type occurred in 68% of patients 10.5% had focal seizures, 5% focal seizures becoming generalised. 15% of our patients presented with status epilepticus.

11 out of the 14 puerperal CVT (78.5%) of the study had seizures, while 8 out of 33 (24.2%) non puerperal had seizures. This suggests that seizures are more common in puerperal CVT. This is supported by higher incidence of seizures in puerperal CVT patients of Nagaraja et al.,[52] Srinivasan and Natarajan et al[64].

Similarly the authors who reported comparitively lower incidence of seizures, also had a very few puerperal CVTs in their series (Bousser et al.,[9] Desichens et al.,[32] and Daif et al[25].

Alteration in level of consciousness (ALC)

About 46.8% of our patients had altered sensorium at the time of presentation. 4 (8.4%) patients were in coma at the time of presentation.

TABLE XIII : (ALC IN VARIOUS SERIES)

Sl.No.	Authors (No. of patients)	Percentage
1.	Srinivasan and Natarajan[63], (90)	44%
2.	Bousser et al.,[9] (38)	26%
3.	Nagaraja et al.,[52] (200)	81%
4.	Cantu and Barinagarementeria.,[55] (113)	61.2%
5.	Nagaraja et al.,[75] (405)	58%
6.	Zhang et al.,[75] (23)	39%
7.	Present study (48)	46.8%

Our study suggests that ALC occurs in major proportion of our patients. Our incidence is similar to that of most other authors except that Bousser et al.,[9] noted ALC in only 26% of cases.

The probable reasons for discrepancy in our study and Bousser et al., 9) may be that :

Seizures were less frequent in their study (29%) while we noted seizures in 40.4% of our patients. Seizures especially in cluster may also contribute to

altered sensorium and this may explain the lesser frequency of ALC in Bousser et al.,[9] series.

Focal deficits

In older series by Kalbag et al.,[43] Krayenbhul et al.,[46] focal deficits such as motor or sensory deficits, dysphasia, cranial nerve deficits occurred in 50 - 75% of patients. Nagaraja et al.,[55] reported focal deficits in 66.4% of their 405 puerperal CVT patients. Daif et al.,[25] reported focal deficits in 32% of 40 patients and Zhang et al.,[75] in 52% of 23 patients.

In our study 55.3% of 47 patients had focal neurological deficits (motor, sensory, cranial nerve deficits).

Hemiparesis was the common focal deficit. It was seen in 57.6% of 25 patients with focal deficit. Among patients with focal deficits 19.2% had monoparesis, 15.3% had multiple cranial nerve deficits. Paraplegia, sensory deficits were uncommon manifestations. It becomes difficult to assess the focal deficits with worsening sensorium

PAPILLEDEMA

Bilateral papilledema was noted in 78.7% of 47 patients.

TABLE - XIV : PAPILLLEDEMA IN VARIOUS SERIES

Sl.No.	Authors (No. of patients)	Papilledema (Percentage)
1.	Srinivasan and Natarajan et al.,[63]	15%
2	Bansal et al.,[6]	35%
3.	Bousser et al.,[9]	45%
4.	Nagaraja et al.,[55]	14%
5.	Cantu et al.,[17]	45%
6.	Daif et al.,[25]	66.7%
8.	Present study	78.7%

Thus the reported incidence of papilledema in CVT ranges from 14 to 80%.

ISOLATED INTRACRANIAL HYPERTENSION.

In Ameri and Bousser et al.,[3] study, 20-40% of patients presented with features of isolated intracranial hypertension with headache, papilledema $\pm$ abducens palsy. 47% of the 40 patients of Daif et al.,[25] presented with isolated ICT.

In our series, 27.6% of 47 patients presented with manifestations of isolated intracranial hypertension.

This reflects the need for use of neuroimaging modalities like MRI with MRA in patients presenting with idiopathic ICT.

CT SCAN FINDINGS

As has been discussed earlier, CT scan with and without contrast injection is the first neuro imaging examination to carry out when CVT in clinically suspected, both to rule out other conditions and try to confirm CVT. A wide variety of abnormalities are seen on CT. The features primary due to thrombosis of veins or sinuses are called direct signs and those due to secondary effects on brain parenchyma are referred to as indirect sign. Though CT is very useful and non - invasive procedure it may be normal in 10 - 20% of cases (Chiras et al.,[20] Rao et al.,[59]).

TABLE - XV : CT SCAN FINDINGS

Sl. No.		Rao et al,[59] (31)	Bousser et al.,[9] (38)	Nagaraja et al.,[53] (68)	Present Study (47)
1.	Cord Sign	6.4%	-	21.9%	8.5%
2.	Empty delta sign	35.5%	12%	32%	6.3%
3.	Non Hemorhagic infarct	9.7%	32%	51.6%	21.2%
4.	Hemorrhagic infact	19.7%	12%	40.9%	36.1%
5.	Intracerebral hematoma	3.2%	-	3.1%	-
6.	SAH	-	-	-	6.3%
7.	SDH	-	-	-	2%
8.	Tentorial enhancement	9.7%	16%	16%	4.2%
9.	Small ventricles	32.3%	52%	-	8.5%
10.	Normal	9.7%	20%	10.9%	21.2%

Normal CT scans are reported in 10 - 20% of patients (Bousser et al.[9], Nagaraja et al.,[53] Rao et al.,[59]). Daif et al.,[25] found 42% of 40 patients had normal scans, whereas in Zhang et al., series[75], 41% had normal scans. In our study, CT scan brain was normal in 21% of the patients.

14.8% of patients of our 47 cases had direct evidence of CVT. 8.5% had cord sign, 6.3% had empty delta sign.

27.6% of patients had indirect evidence of CVT like cerebral edema, tentorial enhancement, obliteration of cistern, squashing of ventricles.

In our study indirect signs were common than direct signs.

Hemorrhagic infarcts were the most common parenchymal abnormality. 36% had hemorrhagic infarct, 19% had non - hemorrhagic infarct. Subdural hemorrhage and sub dural hematoma were rare abnormalities seen in 6.3% and 2% respectively.

Hence though CT scans are very valuable as initial neuro - imaging modality in suspected case of CVT to rule out other lesion (arternal strokes, tumors, abscess) and in some cases making a direct diagnosis of CVT, most of the cases require MR imaging or angiography confirmation of the diagnosis.

MRI FINDINGS

MRI with MRA was done in all 47 patients in our study. It was abnormal in all patients.

TABLE - XVI

Sl. No.		Daif et al.,[25] (n=40)	Bousser[13] (n=135)	ISCVT[38] (n=624)	Present Study (n=47)
1.	Superior Sagittal Sinus	85%	70.3%	62%	74.7%
2.	Transverse Sinus	2%	68.8%	86%	34%
3.	Sigmoid sinus	5%	13.3%	51.6%	36.1%
4.	Internal jugular vein	-	-	12%	8.5%
5.	Straight Sinus	5%	-	-	12.7%
6.	Cavernous sinus	-	2%	-	4.2%
7.	Vein of galen & Deep cerebral veins	-	6%	11%	6.3%
8.	Cortical veins	5%	36%	17%	6.3%

In our study superior sagittal sinus in the most common sinus involved in CVT. 74% of 47 patients had partial or complete occlusion of superior sagittal sinus. It was the only sinus involved in 16 (34%) of patients. This finding in similar to most other studies.

Transverse and sigmoid sinus are the next more common sinus involved in CVT in 34.4% and 36.1% of our patients.

Isolated sinus involvement was seen in 18 (38%) of the patients. 67% patient had multiple sinus thrombosis. Most of the isolated sinus involved was superior sagittal sinus.

63.7% of cases of Bousser et al.,[13] and 65% of patients in Daif et al.,[25] study had multiple dural signs involvement.

PREDIPOSING / RISK FACTORS

In Bousser et al.,[13] predisposing factors were identified in 80% of patients. In 75% patients of 40 patients by Daif et al.,[25] prediposing factors were identified.

In our study, predisposing factors were identified in 66% of our 47 patients. In 34% patients risk factors / etiologic factors contributing to CVT were not identified.

Infective causes were responsible for CVT in 8% of 38 patients in Bousser et al[13]. In Shell and Rathe et al.,[62] study, infection was the cause of CVT in 16% and 17% respectively. 7% of 40 patients of Daif et al.,[25] had infections as risk factor.

In our study 14% of patients had infectious etiology for their CVT. This finding in similar to the incidence in studies by other authors. Mastoditis, sinusitis, TBM, HIV were the inefctious causes our series. Mastoditis was the most frequent infection.

Among the non - infectious etiology, CVT associated with pregnancy and puerperium was the most common cause 29.7% of our 47 patients were puerperal.

Only 1(2.6%) of 38 patients by Bousser et al.,[9] was puerperal and 1 (2.5%) of Daif et al.,[25] was post partum. Indian studies by Nagaraja et al.,[52] Bansal et al.,[6] had found high incidence puerperal CVT in their series.

8.5% of the patients in our study had history of oral contraceptive in take. 27.5% of Deschiens et al.,[32] 7.9% of Bousser et al.,[9] had history of oral contraceptive use. De Bruijn et al.,[27,29] observed an significant increased risk of CVT in patients on oral contraceptives.

4.2% of patients in our study were diagnosed as SLE (one patient had post partum CVT and the other had OCP use as a risk factor).

This observation stresses the need for extensive work up of hereditary thrombophilic conditions even in patients who had one obvious risk factor (like puerperium and OCP intake).

4.2% of study were diagnosed as primary antiphospholipid antibody syndrome and all of them were males.

Cantu et al.,[17] review 67 patients of puerperal and 46 cases of non - puerperal and found Lupus Anticoagulant in 2 (4%) of the non - puerperal patients.

Zuber et al.,[76] estimated aPL in 16 of their 19 CVT patients and found LA in only 1 patient (5%).

In Bousser et al.,[9] study of 38 patients 16% of case had Behcet disease. 25% of the 40 patients in study by Daif et al.,[25] were diagnosed as Behcet's disease. There were no patients of Behcet's disease in our patients.One of our 47 patients in our study had protein C and Protein S deficiency. The contribution of hereditary thrombophilic conditions as a risk factor could not be estimated because these investigations were done only in very few patients. This suggest that the number of patients with hereditary thrombophilic conditions could be higher and reflects the necessity of these investigations in all patients with CVT.

TREATMENT

Einhaupl et al.,[36] in his study of 71 patients showed significant benefit with heparin even in patients with hemorrhagic infarcts.

Diaz et al.,[33] reviewed 203 CVT patients reported between 1942 and 1990 and compared the outcome of patients treated (149 patients) and not treated with heparin. 91% survived in the first group compared to 36% in second group.

Nagaraja et al.,[54] compared the effect of intravenous unfractionated heparin with that of placebo in 57 patients of puerperal CVT and showed non - significant benefit of anti-coagulant treatment as compared with placebo.

In our study, all 47 patients received anticoagulation with heparin until the patient stabilized, even if the patient had hemorrhagic infarct, sub-arachnoid hemorrhage, subdural hematoma. Once stabilized, patients were changed to oral anti - coagulants. If no prothrombotic risk factors were identified, anti-coagulants were stopped after 6 months. Patients with predisposing thrombophilic conditions were put on long term oral anti-coagulation. There was no deterioration in the clinical neurologic status in treating patients with hemorrhagic infarcts, with heparin and most of the patients in proved. Hence we recommend anticoagulation with heparin in all CVT patients in acute stage, even the presence of hemorrhagic infarct, ICH and SAH provided there are no other contra-indication to its use.

TABLE XVII CASE FATALITY IN RECENT SERIES OF CVT

Sl.No.	Authors (No. of patients)	Mortality (%)
1.	Einhaupl, et al.,[38] (71)	14%
2	Ameri, et al.,[3] (110)	6%
3.	Barinagarrementeria et al.,[17] (78)	23%
4.	Daif et al.,[25] (40)	10%
5.	De Bruijn et al.,[30] (59)	10%
6.	Ferro, et al.,[40] (142)	6%
7.	Breteau et al.,[14] (55)	4%
8.	ISCVT[38] (624)	4%

	Pregnancy or Puerpericum	
9.	Cantu et al.,[17] (67)	9%
10.	Nagaraja et al.,[58] (150)	17%
11.	ISCVT[38] (77)	4%
12.	Present Study (47)	4.5%

Mortality rate in our study in similar compared to experience of authors from recent studies.

At the end of 4 weeks, 72% of our 47 patients recovered completely. 23.4% had residual neurological sequelae like residual focal deficits, seizures and visual loss.

OUTCOME IN RECENT SERIES OF PATIENTS WITH CVT

Outcome	**Einhaupl et al.,[36] (n=71)**	**Bousser et al.,[13] (n=135)**	**Daif et al., [25](n=40)**	**Cantu and BarinagarrementeriaFet al.,[17]**		**Present Study (n=47)**
				(P)67	**(NP)46**	
Total recovery	42(59%)	101(75%)	29(72%)	36 (33%)	24 (52%)	34 (72%)
Minor Sequelae	13(18.5%)	16(12%)	3(3%)	19 (28.3 %)	3(6.5%)	11(23.4%)
Major Sequelae	6(8.5%)	12(8%)	4(10%)	6(9%)	4(9%)	
Death	10(14%)	6(5%)	4(10%)	6(9%)	15(33%)	2(4.2%)

The short term outcome our in study was similar to other studies.

72% had good neurologic outcome.

Since the patients were not followed up for a long duration, we could not comment on the long term outcome out come of patients. But unlike arterial strokes, if the patient survives the early period, CVT has got a relatively good prognosis.

CONCLUSION

1. CVT is far more common neurologic problem than previously assumed.

2. The mode of onset is variable and spectrum of clinical presentation is wide.

3. Diagnosis of CVT requires high index of suspicion.

4. MRI with MRA is the most sensitive neuro-imaging modality in diagnosis of CVT.

5. All patients with isolated ICT should undergo MRI with MRA to rule out CVT.

6. Demonstration of underlying etiology may be difficult of when the cause is not clinically evident.

7. Multiple risk factors can be present in a single patient.

8. Systematic workup for hereditary thrombophilic conditions should be undertaken in every patient ,even when they have one obvious risk/etiologic factor of CVT.

SUMMARY

The study involved 47 patients of proven cases of CVT.

The clinical profile, neuroimaging findings, predisposing / etiologic factors, treatment and outcome at 4 weeks were studied.

Mean age of study cohorts was 28.5 years. 51% were males, 49% females. Most of them (70.2%) had subacute onset, 23% had acute, 6.7% chronic onset of symptoms.

29.7% were puerperal CVTs and 60.8% were non puerperal. Puerperal CVT had earlier mean age of onset. (25.1 years) compared to non - puerperal CVT (31.7 years). Also most of puerperal CVT patients (57%) had acute onset where as most of non - puerperal CVT patients (81.8%) had subacute onset symptoms.

Headache is the most common clinical presentation in 85% of cases. Other clinical features were seizures (40.4%), alteration in consiousness (46.8%), focal deficits (55.3%), bilateral papilledema (78.7%). (27.6%) of patients presented with isolated ICT.

Of the seizures, GTCS was most common (68.4%). Others were focal (10.5%), focal with secondary generalisation (5.2%) and status epilepticus (15.7%).

CT was normal in 21% of study patients. Indirect signs (27.6%) were common than direct signs (14.8%).

MRI with MRA was reliable as sole investigation in the diagnosis of CVT. (38.2%) had isolated sinus involvement (61.7%) had multiple sinus involvement. SSS was the most commonly involved sinus.

Other commonly involved sinuses are sigmoid (36.1%) and lateral sinus (34%). (10.6%) had cortical vein involvement deep vein system was involved in (6%) and cavernous sinus in 4.2% and IJV in (4.2%).

Predisposing / etiologic factors identified (66%) of our patients. Varied risk factors were identified in our patients. (14.5%) were related to infectious causes. Among the non infectious etiology puerperuim (29.7%) was the common risk factor. Others include OCP use (8.5%), SLE (4.2%), APLA (4.2%).

All our patients improved with heparin treatment despite hemorrhagic and SAH.

Mortality was (4.2%) in out study. Most of our patients had a good neurological outcome. (72%) had complete recovery and (23%) had residual sequelae like focal deficits, recurrent seizures and visual loss.

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PROFORMA

NAME : **AGE :** **SEX :**

OCCUPATION : **INCOME :** **MRD NO :**

D.O.A. **D.O.D.** **HISTORY:**

PRESENTING SYMPTOMS:

a) NEUROLOGIC

SYMPTOMS	**PRESENT/ABSENT**	**DURATION**

Headache :

Type :

Vomiting :

Loss of consiousness:

Altered sensorium :

Convulsions :

No:of episodes :
Type
Focal
GTCS
Simple partial
Complex partial
Status epilepticus

Limb weakness : Right/left Upper limbs Lower limbs

Speech disturbances :

Neuro psychiatric manifestations :

Chronology of symptoms(short history):

What is the progression of illness till interview?
Static
Progressive
Regressive
Fluctuating

NON-NEUROLOGIC SYMPTOMS: Present or Absent

Fever
Sinusitis
Ear ache
Ear discharge
Diarrhoea
Rhematologic/musulo skeletal symptoms:
Arthralgia
Oral ulcers
Alopecia
Skin rash
Photo sensitivity
Raynauds phenomenon
Others(specify)

PAST HISTORY :

DM
HT
CAD/valvular
TB
Seizure disorder
Migraine
Thyroid disorders(hyper/hypo)
Others(specify)

DRUG HISTORY:

OCP
Anticoagulants
Others

FOR FEMALES:

MENSTRUAL HISTORY
Regularity
LMP
OCP intake
Marriage :
Consanguinity:

OBSTETRIC HISTORY

Gravida Para
Recurrent abortions:
H/O PIH/ecclampsia

PUERPERAL/NATAL HISTORY:

Date of delivery
Normal vaginal delivery/ LSCS:
Complications during labor
H /o DVT

FAMILY HISTORY:

DM
HT
CAD
STROKE
CONVULSIONS
CONNECTIVE TISSUE DISORDERS

PERSONAL HISTORY

SMOKING
ALCOHOL
DRUG ADDICTIONS/ABUSE

EXAMINATION : Present or Absent

Febrile :
Anemia :
Clubbing :
Edema :
Jaundice
Cyanosis :
Lymphadenopathy :
Signs of dehydration
Signs of peripheral thromophlebitis

PR
BP

CNS EXAMINATION:

Higher functions:
Consiousness
Normal
Drowsy
Stupor
Coma
GCS
Speech
Other Hf abnormality (Normal/Abnormal)
(Present / Absent)

CRANIAL NERVES	RIGHT	LEFT	NORMAL / ABNORMAL
I Olfaction			
II Visual acuity Field Color vision			
III, IV, VI pupil size Ocular movts Palsy III/IV VI			
V			
VII-Facial lag UMN/LMN			
VIII			
IX			
X			
XI			
XII			

SPINO MOTOR SYSTEM:

	RIGHT	LEFT	NORMAL / ABNORMAL
Tone UL LL			
Power UL LL			
Reflexes			
Superficial corneal Conjuctival Abdomen plantar			
Deep tendon reflexes UL LL			
plantar			
Co-ordination			

SENSORY SYSTEM

Touch/pain/vibration/JPS UL LL		

FUNDUS

Papilledema HT retinopathy Dm retinopathy Retinal hemorrhage Optic atrophy		

Present or Absent

CEREBELLAR INVOLVEMENT
EXTRA PYRAMIDAL INVOLVEMENT
BLADDER/AUTONOMIC INVOLVEMENT
MENINGEAL SIGNS
Neck rigidity
Kernigs

OTHER SYSTEMS:

ENT EXAMINATION Sinusitis : present/absent

Mastoids : present/absent

CVS - Normal/Abnormal

ABNORMAL FINDINGS

RS : Normal / Abnormal

ABDOMEN EXAMINATION : Normal/Abnormal

CLINICAL DIAGNOSIS:

INVESTIGATIONS: DONE / NOT DONE NORMAL OR ABNORMAL

1) Hemogram :
   Hb
   TC
   DC
   ESR
   Platelets

2) Bl. Sugar:
   urea
   creatinine
   electrolytes

3) Lipid profile:
   Total cholesterol
   TGL
   LDL

4) BT
   CT
   aPTT
   PT/INR

5) VDRL

6) HIV

7) X-ray chest

8) ECG

9) ECHO

10) APLA

11) LAC

12) PROTEIN C

13) PROTEIN S

14) FACTOR V LEIDEN

15) ANTI THROMBIM III

16) SE.HOMOCYSTEINE

17) ANA

18) RHEUMATOID FACTOR

19) USG ABDOMEN

20) DOPPLER LOWER LIMBS

21) OTHERS(SPECIFY)

22) CT BRAIN: (with and without - contrast)

	Present or Absent
Normal	
Non - hemorrhagic infarct	
Hemorrhagic infarct	
Intrancerebal hematoma	
Sub-arachnoid hemorrhage	
Sub-dural hematoma	
Direct Signs	
Cord Sign	
Empty delta sign	
Indirect signs	
Cerebral edema	
Obliteration of cistern	
Ventricle size	
a. Squashed	
Tentorial enhancement	
Gyral enhancement	
Falx enhancement	

23) MRI BRAIN &MRV:

	Involved / Not Involved or present/absent.
Sinus Involvement	
Superior sagittal sinus	
Inferior sagital sinus	
Transverse sinus	
Sigmoid sinus	
Cavernous sinus	
Straight sinus	
Internal jugular veins	
Cortical vein thrombosis	
Deep cerebral vein involvement	
Non hemorrhagic infarct	
Hemorrhagic infarct	
Sub-arachnoid hemorrhage	
Sub dural hematoma	

TREATMENT: **GIVEN / NOTGIVEN**

1**)** **HEPARINS**:

TYPE
DOSE
DURATION

2) ORAL ANTICOAGULANTS GIVEN/NOT GIVEN

DOSE
DURATION

3) ANTI EDEMA MEASURES GIVEN/NOT GIVEN

MANNITOL
FRUSEMIDE
STEROIDS

4) ANTICONVULSANTS GIVEN/NOT GIVEN

DOSE
DURATION

PROGRESS (AT 4 WEEKS) :

COMPLETE RECOVERY
RESIDUAL DEFICIT
DEATH

FOLLOW UP:

RISK FACTORS OF CEREBRAL VENOUS THROMBOSIS

IDENTIFIED / NOT IDENTIFIED :

INFECTIOUS / NON INFECTIOUS :

SPECIFY :

ABBREVIATIONS

CVT	:	Cerebral venous thrombosis
CTD	:	Connective tissue disorders
DM	:	Diabetes mellitus
HT	:	Hypertension
CAD	:	Coronary artery disease
SSS	:	Superior sagittal sinus
LS	:	Lateral sinus
TS	:	Transverse Sinus
SS	:	Sigmoid sinus
STS	:	Straight sinus
IJV	:	Internal jugular vein
DVS	:	Deep Venous system
Corti.v	:	Cortical vein
CS	:	Cavernous sinus
P	:	Puerperal
NP	:	Non - Puerperal
MRI	:	Magnetic Resonance Imaging
MRA	:	Magnetic resonance angiogram
MRV	:	MR Venogram
APLA	:	Antiphospholipid antibody syndrome
SLE	:	Systemic lupus Erythematosus
ICT	:	Intracranial tension
ICP	:	Intracranial pressure
GTCS	:	Generalised tonic clonic seizures
St. Epi	:	Status Epilepticus
CT	:	Computer Tomography
HI	:	Hemorrhagic infarct
NHI	:	Non - Hemorrhagic infarct
ICH	:	Intracranial hemorrhage

SAH	:	Sub-arachnoid hemorrhage
SDH	:	Sub- dural hemorrhage
CSF	:	Cerebrospinal fluid
ESR	:	Erythrocyte Sedimentation rate
PMN	:	Polymorphonuclear cell
ALC	:	Alteration in level of consiousness
BPE	:	Bilateral paplledema
LAC	:	Lupus anti-coagulant
OCP	:	Oral contraceptives
SA	:	Sub-acute
Chr.	:	Chronic
HP	:	Hemiparesis / hemiplegia
M	:	Monoparesis / monoplegia
P.P	:	Paraparesis / paraplegia
Cr.N. Defi.	:	Cranial Nerve deficitis
Men. Irrt.	:	Meningeal irritation
C.Ede.	:	Cerebral edema
Obli. Cis.	:	Obliteration of Cisterns
Vent. Squ	:	Ventricles squashed
Tent. Enh.	:	Tentorial Enhancement
Gy / Fx. Enh.	:	Gyrac or Falx enhancement
Inf.	:	Infectious etiology
UL	:	Upper limb.
LL	:	Lower limb
JPS	:	Joint position sense
CVS	:	Cardiovascular system.
RS	:	Respiratory system
DVT	:	Deep vein thrombosis
PIH	:	Pregnancy induced hypertension

Sl. No.	Age	Sex	P/NP	On Set			Head. ache	Seiz. ures	Type of Seizures				Focal Deficits					PE	Men. Irrit.	Iso. ICT	Consi. ous	ALC	Level of Consiousn	
				Acute	S.A	Chr.			Focal	Sec. Gen	St. EPL	GTCS	Sen. sory	HP	M	PP	Cr. N. Defic.						Dro. wsy	Stu. por
1	18	F	P	-	+	-	+	+	-	-	-	+	-	-	+	-	-	+	-	-	-	+	-	-
2	26	F	P	+		-	+	-	-	-	-	-	+	-	+	-	-	+	-	-	+	-	-	-
3	27	F	P	-	+	-	+	+	-	-	-	+	-	-	-	-	-	+	-	-	-	+	-	+
4	20	F	P	+		-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-
5	22	F	P	-	+	-	-	+	-	-	+	-	-	-	-	+	-	+	-	+	-	+	+	-
6	25	F	P	-	+	-	+	+	-	-	+	-	-	-	-	-	-	+	-	-	-	+	+	-
7	29	F	P	+		-	+	+	-	+	-	+	-	-	+	-	-	+	-	-	+	-	-	-
8	24	F	P	+		-	+	+	-	-	+	-	-	-	-	-	-	+	-	-	-	+	-	+
9	24	F	P	+		-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+
10	36	F	P	+		-	-	+	-	-	-	+	-	-	+	-	-	+	-	-	-	+	-	+
11	22	F	P	-	+	-	+	+	-	-	-	+	-	-	-	-	-	+	-	-	-	+	+	-
12	27	F	P	-	+	-	+	+	-	-	-	+	-	+	-	-	-	-	-	-	-	+	+	-
13	26	F	P	+		-	+	+	-	-	-	+	-	+	-	-	-	+	-	-	-	+	+	-
14	26	F	P	+		-	+	+	+	-	-	-	-	-	-	-	+	+	-	-	+	-	-	-
15	35	F	NP	-	+	-	+	-	-	-	-	-	-	+	-	-	-	+	-	-	+	-	-	-
16	23	F	NP	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-
17	26	F	NP	+		-	+	-	-	-	-	-	-	-	-	-	+	+	-	-	-	+	+	-
18	24	F	NP	-	+	-	+	+	-	-	-	+	-	-	-	-	-	+	-	+	+	-	-	-
19	25	F	NP	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-
20	21	F	NP	-	+	-	+	-	-	-	-	-	-	-	-	-	-	+	-	+	+	-	-	-
21	38	F	NP	-	+	-	+	-	-	-	-	-	-	+	-	-	-	+	-	-	+	-	-	-
22	37	F	NP	-	+	-	+	-	-	-	-	-	-	+	-	-	-	+	-	-	+	-	-	-
				On Set					Type of Seizures				Focal Deficits										Level of Consiousn	

Sl. No.	Age	Sex	P/NP	Acute	S.A	Chr.	Head. ache	Seiz. ures	Focal	F with Sec. Gen	St. EPL	GTCS	Sen. sory	HP	M	PP	Cr. N. Defic.	PE	Men. Irrit.	Iso. ICT	Consi. ous	ALC	Dro. wsy	Stu. por
23	27	F	NP	-	+	-	+	+	-	-	-	+	-	+	-	-	-	+	-	-	+	-	+	-
24	22	M	NP	+	-	-	+	+	+	-	-	-	-	+	-	-	-	+	-	-	-	+	-	+
25	22	M	NP	-	+	-	+	-	-	-	-	-	-	+	-	-	-	+	-	-	+	-	-	-
26	38	M	NP	-	+	-	+	-	-	-	-	-	-	+	-	-	-	+	-	-	-	+	-	-
27	29	M	NP	-	+	-	+	-	-	-	-	-	-	+	-	-	+	-	-	-	+	-	-	-
28	48	M	NP	-	+	-	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+
29	27	M	NP	-	+	-	+	-	-	-	-	-	-	+	-	-	+	-	-	-	-	+	-	+
30	38	M	NP	-	-	+	+	-	-	-	-	-	-	-	-	-	-	+	+	-	+	-	-	-
31	38	M	NP	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-
32	32	M	NP	+	-	-	+	-	-	-	-	-	-	+	-	-	-	+	-	-	-	+	-	+
33	27	M	NP	-	+	-	+	-	-	-	-	-	-	-	-	-	-	+	+	-	+	-	-	-
34	27	M	NP	-	-	+	+	-	-	-	-	+	-	-	-	-	-	+	+	-	+	-	-	-
35	36	M	NP	-	-	+	+	+	-	-	-	-	-	+	-	-	-	+	-	-	-	+	+	-
36	55	M	NP	-	+	-	+	-	-	-	-	+	-	-	-	-	-	-	-	-	+	-	-	-
37	55	M	NP	-	+	-	+	+	-	-	-	-	-	-	+	-	-	+	-	-	-	+	+	-
38	24	M	NP	-	+	-	+	-	-	-	-	-	-	-	-	-	-	+	+	-	+	-	-	-
39	22	M	NP	-	+	-	+	-	-	-	-	-	-	-	-	-	-	+	+	-	+	-	-	-
40	25	M	NP	-	+	-	+	-	-	-	-	-	-	-	-	-	-	+	+	-	+	-	-	-
41	26	M	NP	-	+	-	+	-	-	-	-	-	-	-	-	-	-	+	+	-	+	-	-	-
42	35	M	NP	-	+	-	+	-	-	-	-	+	-	-	-	-	-	+	+	-	+	-	-	-
43	25	M	NP	-	+	-	+	+	-	-	-	-	-	-	-	-	-	+	-	-	-	+	+	-
44	33	M	NP	-	+	-	+	-	-	-	-	+	-	+	-	-	-	+	-	-	+	-	-	-
45	34	M	NP	-	+	-	+	+	-	-	-	-	-	+	-	-	-	+	+	+	+	-	-	-
46	64	M	NP	-	+	-	+	-	-	-	-	-	-	-	-	-	-	+	+	-	+	-	+	-
47	34	M	NP	-	+	-	+	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-

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	CT Findings											MRI Findings												Risk Factors Identification									Outcome		
Sl No.	N	NHI	HI	ICH / SAH SDH	Cord. Sign	Empty. Delta	C. ede.	Obli. Cis.	Vent. Squ.	Tent. Enh.	Gy / Fx. Enh.	SSS	ISS	TS	SS	St. S	IJV	Cor. V.	DVS	HI	NHI	ICH / SDH / SAH	Isol. Sinus	Mult. Sinus	ID	UN-ID	Inf.	P	APL	Pr. C	Pr. S	OCP	SLE	Death	Tot. Rec	Res. Def.
1	-	-	+	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	+	-	-	-	-	-	-	+	-
2	-	+	-	SAH	-	-	-	-	-	-	-	+	-	+	-	-	-	-	-	-	+	SAH	-	+	-	-	-	+	-	-	-	-	-	-	+	-
3	-	-	+	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	+	-	-	-	-	-	-	+	-
4	-	-	-	SAH	-	-	-	-	-	-	-	-	-	+	+	-	-	-	-	-	-	SAH	-	+	-	-	-	+	-	-	-	-	-	-	+	-
5	-	-	+	-	+	+	-	-	-	-	-	+	-	-	+	-	-	-	-	+	-	-	-	+	-	-	-	+	-	-	-	-	+	-	-	+
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14	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	+	-	-	-	-	-	-	+	-
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17	-	-	-	-	-	+	-	+	+	-	-	+	-	+	+	-	+	+	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	+	-
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21	-	-	-	SDH	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	-	-	SDH	-	+	-	-	-	-	-	-	-	-	-	-	-	+
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24	-	-	+	-	+	-	-	-	-	-	-	+	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	+	-	-	-	-	-	+
	CT Findings											MRI Findings													Risk Factors Identification									Outcome		
SI No.	N	NHI	HI	ICH / SAH SDH	Cord. Sign	Empty. Delta	C. ede.	Obli. Cis.	Vent. Squ.	Tent. Enh.	Gy / Fx. Enh.	SSS	ISS	TS	SS	St. S	IJV	Cor. V.	DVS	HI	NHI	ICH / SDH / SAH	Isol. Sinus	Mult. Sinus	ID	UN-ID	Inf.	P	APL	Pr. C	Pr. S	OCP	SLE	Death	Tot. Rec	Res. Def.
25	-	-	+	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+
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38	+	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	+	-
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40	+	-	-	-	-	-	-	-	-	-	-	+	-	+	+	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	+	-
41	+	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	+	-
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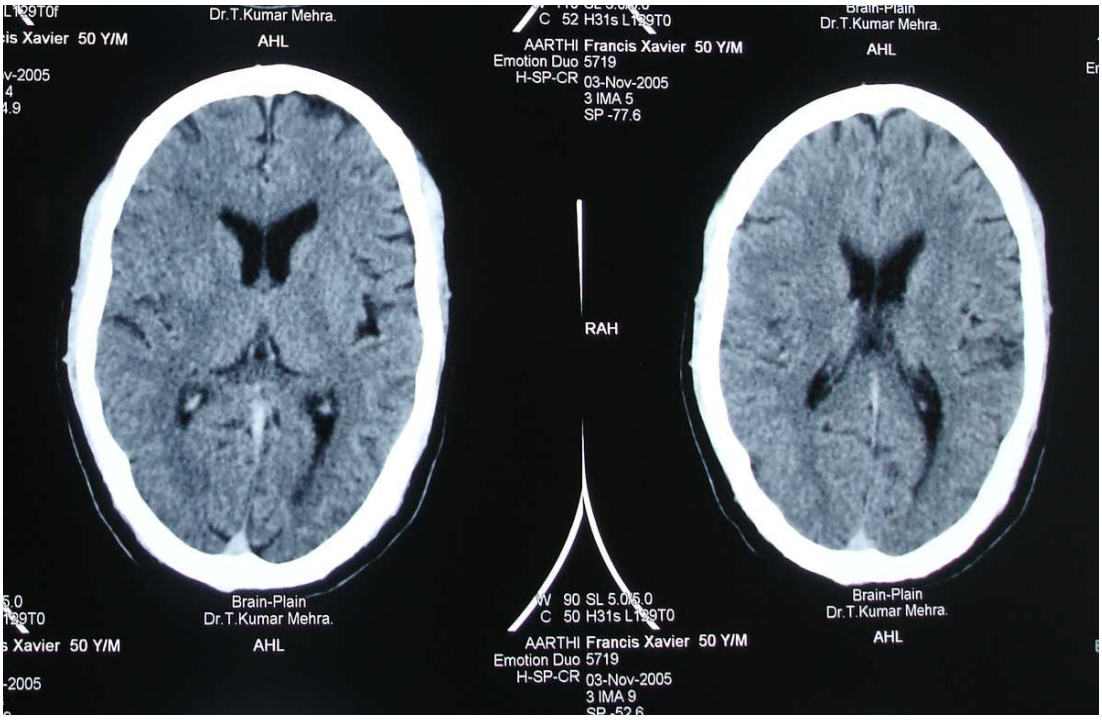
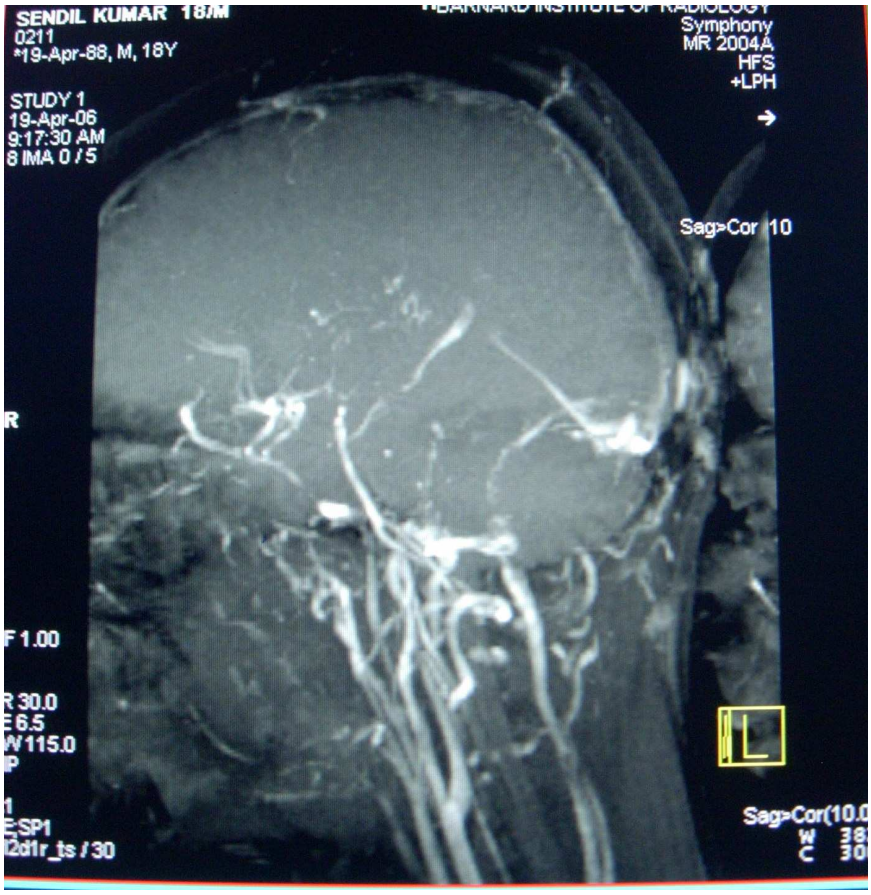


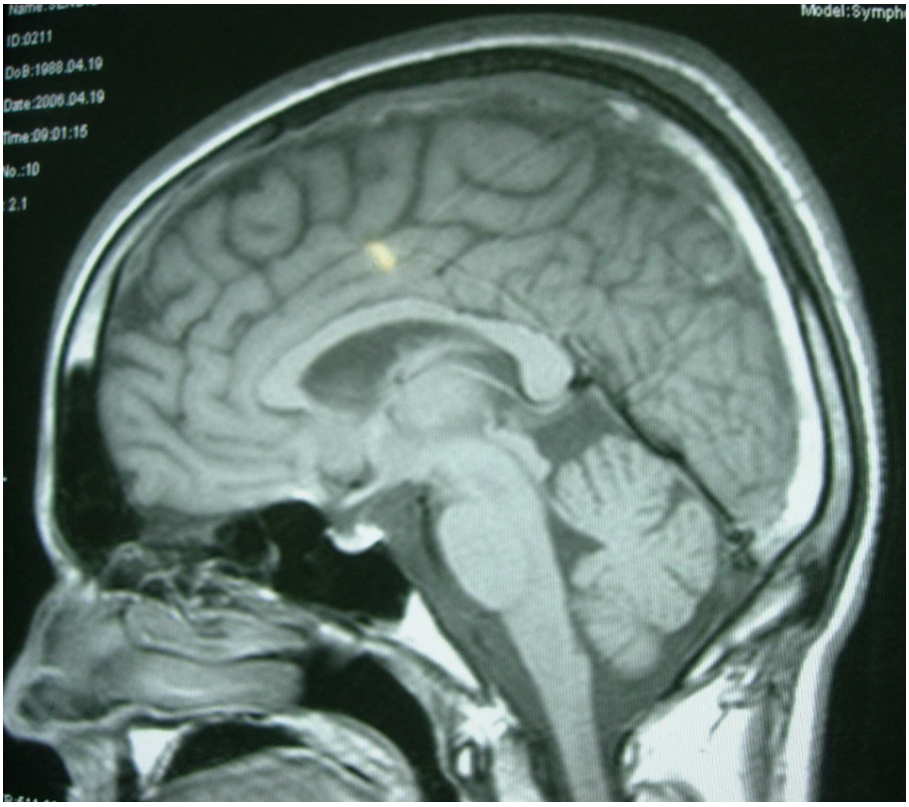
CT Brain plain of a study patient showing the "cord sign" and "dense triangle" of SSS thrombosis

MRI Brain of a CVT Patient T1W image showing bilateral hemorrhagic infarct in parietal region

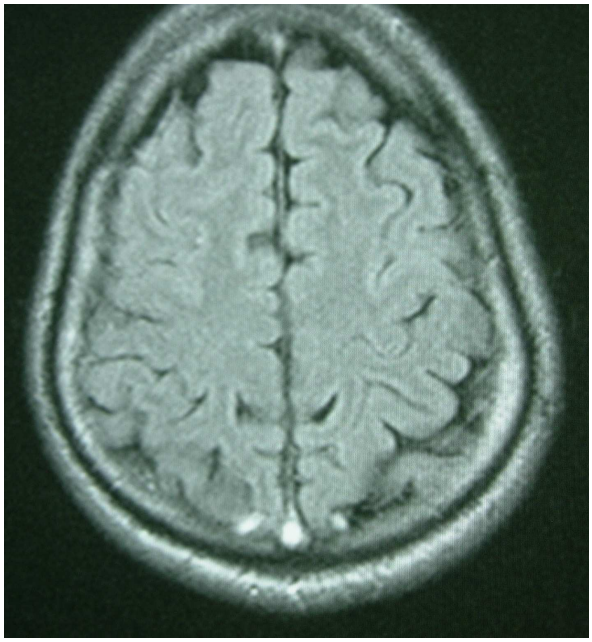


MRV of a study patient showing absence o f flow void in SSS

MRV of a CVT patient showing absence of flow void in the left transverse and sigmoid sinus.



MRI brain sagittal section T1W image showing thrombosed SSS



MRI brain T1W image showing SSS thrombosed and associated cortical vein thrombosis